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Thermoregulatory Control Following Dynamic Exercise

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Post-exercise thermoregulatory control in humans has received limited attention. In the post-exercise period skin blood flow and sweating return to pre-exercise levels despite a persistent elevation in esophageal temperature, suggesting an alteration in thermoregulatory control. The esophageal temperature response post-exercise appears to be correlated to the marked cardiovascular changes that occur after dynamic exercise. Recent work has shown that non-thermoregulatory factors associated with hemodynamic changes and hydration status post-exercise may influence the regulation of core temperature during exercise recovery. This review will characterize the thermal response and describe our current understanding of the physiological influences on thermoregulatory control during recovery from dynamic exercise.

Keywords: post-exercise hypotension, heat stress, hypohydration, exercise recovery, non-thermal reflexes.

THE VAST MAJORITY of research on human thermoregulation has been performed on subjects at rest by passive heating or conducted via endogenous heating during dynamic exercise. Relatively less attention has been directed toward thermoregulatory control post-exercise. There is increasing evidence that the thermoregulatory responses observed during the recovery period from dynamic exercise are inconsistent with those seen at rest under exogenous heating conditions or by endogenous heating during exercise. Effective thermoregulation during exercise recovery is particularly important in those individuals performing intermittent bouts of dynamic exercise. Thus, research on the physiology of thermoregulatory responses post-exercise is of importance in athletic, industrial, and military settings. Furthermore, advancing our knowledge of the mechanisms governing post-exercise body temperature regulation will enhance our understanding of such issues as exertional heat stress. For example, the observed attenuation in heat loss post-exercise may exacerbate the risk and/or occurrence of heat-related illnesses, especially in those individuals performing repeated episodes of work in hot environments. This review will describe the current state of research on post-exercise thermoregulation in humans by describing the typical observations during exercise recovery and the possible non-thermal mechanisms contributing to the responses. It should be noted that the majority of the studies discussed in this review have been conducted using short duration bouts of moderate intensity dy-

namic exercise where recovery occurred in thermoneutral conditions unless stated otherwise. While this area of research may provide insight into other related areas of heat stress and exercise, this review will not examine the vast number of studies examining exertional heat injury, heat stroke, and other similar investigations of severely hyperthermic individuals. These topics have been reviewed previously.

Post-Exercise Temperature Response

Metabolic heat production during dynamic exercise results in body heat storage and thus an increase in internal temperature, although the individual is generally prevented from reaching severe hyperthermia by initiating the cooling responses of skin blood flow and sweating. During the exercise recovery period the stored body heat must also be removed by the thermoregulatory mechanisms of skin blood flow and sweating, which tend to promote heat dissipation. However, during inactive recovery, esophageal temperature remains elevated and skin blood flow and sweating are markedly reduced. Thus, there is a reduction in the rate of heat loss. The observed attenuation in heat loss post-exercise may be secondary to such factors as nonthermal physiological mechanisms, and can be further compromised under hot environmental conditions. This section will characterize the nature of core and muscle temperature responses during recovery as well as the responses of skin blood flow and sweating.

Esophageal temperature response: The primary mechanism for stimulating heat loss responses is via a hypo-

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thalamic feedback loop that receives an integrated signal from skin and core temperature (50). It is not possible to measure hypothalamic temperature in human studies and thus esophageal temperature is used a surrogate 'core temperature.' Esophageal temperature is representative of arterial blood temperature at the level of the heart and is commonly measured in thermoregulatory studies by inserting a catheter-like thermocouple into the esophagus nasally to the level of the heart (65). This review will, therefore, use the term esophageal temperature, which is an index of hypothalamic temperature in thermoregulation research.

Recent studies have shown that esophageal temperature remains elevated for a prolonged period post-exercise (34,35,68,70). The disturbance in post-exercise thermal homeostasis was first characterized by Thoden et al., who observed that esophageal temperature remains elevated by approximately 0.4–0.5°C (68). Although several studies have reported a post-exercise temperature profile similar to their observations, none specifically addressed the extended elevation in esophageal temperature (1,2,16,59,70). Thus, pre-exercise body temperature is not re-established post-exercise and is contrary to what set-point theory would predict (22). Based on the set-point control theory of human thermoregulation, one would surmise a rapid reestablishment of esophageal temperature in response to the absence of the heat-producing events of exercise to pre-exercise levels. Of note, the sustained esophageal temperature elevation recorded by Thoden et al. (68) was not due to an increased rate of heat production as oxygen consumption had returned to baseline values within 5–10 min of exercise termination. This is in contrast to the work of Williams et al. (71), who observed sustained elevations in oxygen consumption up to 135-min post-exercise. These observations might be due to protocol differences. In the study by Thoden et al. (68), subjects performed 15 min of treadmill exercise at 70% $\dot{V}O_{2peak}$, while participants in the study by Williams and colleagues performed 60 min of upright cycling exercise at 60% $\dot{V}O_{2peak}$. Nevertheless, the possible heat gain associated with a sustained elevation of post-exercise oxygen consumption is not sufficient to maintain esophageal temperature at levels of 0.4–0.5°C above pre-exercise values.

Kenny et al. (36) subsequently demonstrated that the reported disturbance in thermoregulatory control was likely due to a residual effect of exercise per se and not due to an increase in body heat content alone. In their study, subjects were heated by whole-body immersion in warm water (42°C), which produced a similar rate of rise in esophageal temperature and end-immersion temperature comparable to that of 15 min of moderate intensity exercise. Specifically, no sustained increase in esophageal temperature was measured following whole-body exogenous heating.

One of the most striking observations during exercise recovery stems from the fact that sweating (41), skin blood flow, and skin temperature (41,70) return to pre-exercise values despite a persistent elevation in esophageal temperature (Fig. 1). Based on set-point theory (22), one would predict increases in skin blood flow and

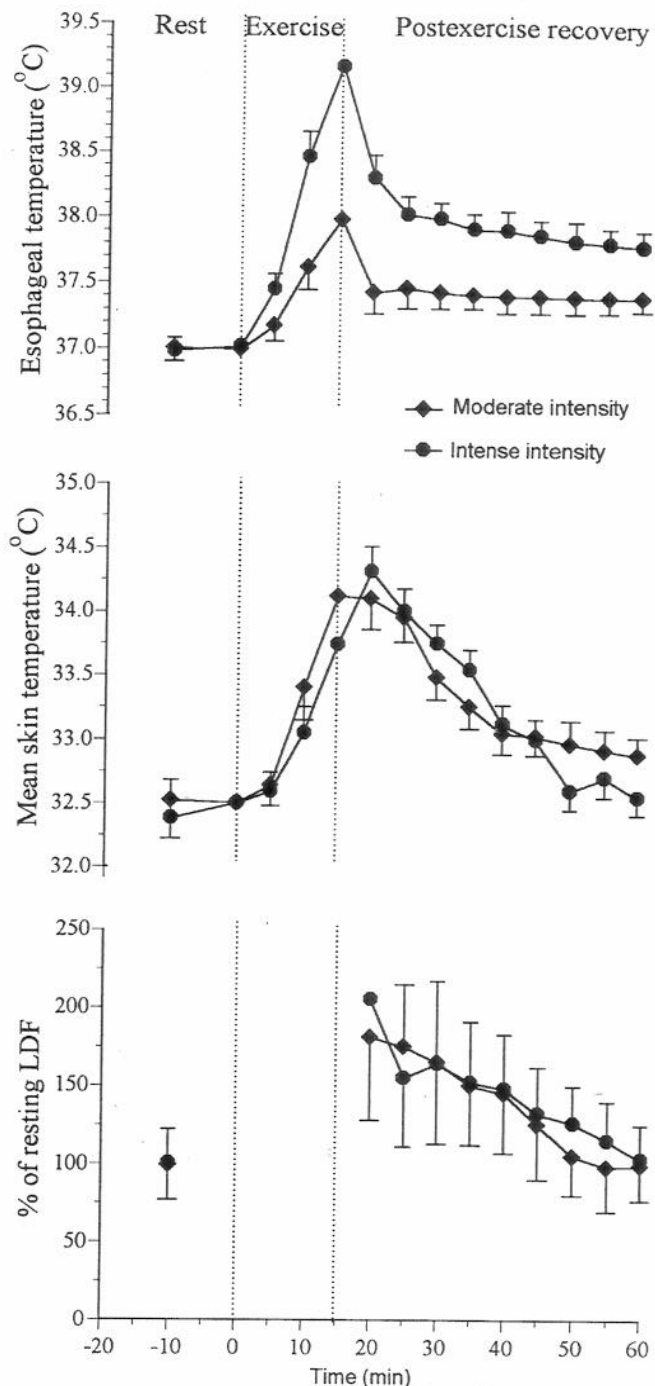


Fig. 1. Typical skin blood flow, esophageal and skin temperature responses during exercise recovery. Mean (\pm SD) esophageal temperature (top), skin temperature (middle), and skin blood flow (bottom) during baseline resting, exercise (except skin blood flow), and post-exercise recovery periods for the two 15-min treadmill exercise conditions ($n = 8$): moderate exercise (70% of maximum oxygen consumption, $\dot{V}O_{2peak}$) and intense exercise (93% $\dot{V}O_{2peak}$). LDF: laser Doppler flowmetry. From Ref. 41; used with permission

sweating proportional to increases in esophageal temperature via a hypothalamic negative feedback loop. However, despite an elevation in esophageal temperature, which is known to be one of the prime drivers of heat loss responses, skin blood flow and sweating return to pre-exercise values. This suggests a possible resetting of the skin blood flow/sweating-esophageal

temperature relationship post-exercise (Fig. 2 and 3). If such a relationship was not altered post-exercise, one would expect that such increases in skin blood flow and sweating would serve to enhance heat dissipation, therefore increasing the rate of heat loss with a resultant decay of esophageal temperature back to baseline. On the contrary, reduced skin blood flow and sweating responses and a prolonged elevation in esophageal temperature are typically observed.

Muscle temperature response: In an effort to better understand the possible mechanisms for the prolonged esophageal temperature elevation, Kenny et al. (45) studied post-exercise tissue temperature responses. They demonstrated that the post-exercise elevation in esophageal temperature is paralleled by a sustained increase in muscle temperature. Specifically, esophageal temperature showed a rapid decrease in the first minutes of exercise recovery followed by a prolonged, sustained elevation of $\sim 0.3^{\circ}\text{C}$. A similar pattern of response was noted with muscle temperature. End exercise muscle temperature was $\sim 1.0^{\circ}\text{C}$ higher than the value observed for esophageal temperature. However, at cessation of exercise, active muscle temperature decreased rapidly during the early stages of exercise recovery to achieve values equal to that of esophageal temperature (~ 20 min post-exercise). Subsequently deep muscle temperature remained relatively unchanged from esophageal temperature for the duration of the 60-min recovery. Their observations provide evidence to suggest that the post-exercise esophageal temperature response and, therefore, the rate of esophageal temperature decay, is significantly influenced by convective heat transfer between muscle and core.

As described later in this review, a consequence of dynamic exercise includes post-exercise hemodynamic changes which are further exacerbated by an upright, seated recovery posture. Recently it has been shown that an increase in the post-exercise hypotensive response is paralleled by an increase in the magnitude and recovery time of esophageal and active muscle temperatures (38). This effect, in conjunction with a

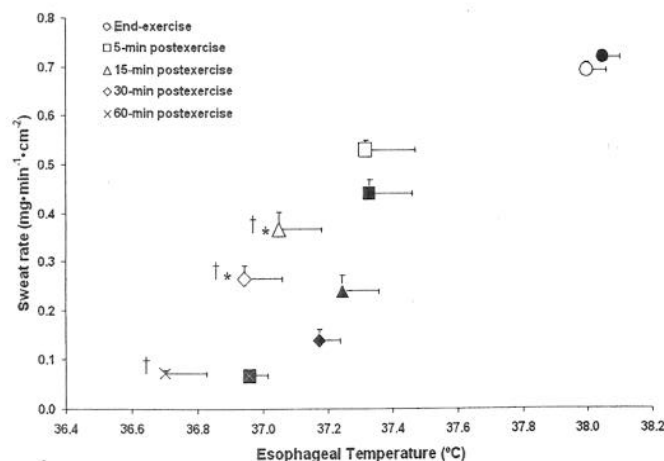


Fig. 2. The relationship of esophageal temperature with sweat rate during recovery from 15 min of cycle ergometry at 75% $\dot{V}O_{2\text{peak}}$ in the upright seated posture (filled symbols) and 15° HDT position (clear symbols). A significant difference between recovery positions at a given time point is denoted by an asterisk (*) for sweat rate and a cross (†) for esophageal temperature. From Ref 49; used with permission.

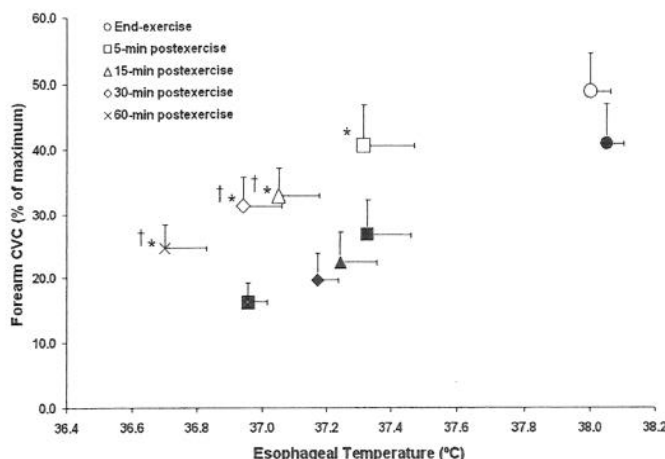


Fig. 3. The relationship of esophageal temperature with cutaneous vascular conductance during recovery from 15 min of cycle ergometry at 75% $\dot{V}O_{2\text{peak}}$ in the upright seated posture (filled symbols) and 15° HDT position (clear symbols). A significant difference between recovery positions at a given time point is denoted by an asterisk (*) for cutaneous vascular conductance and a cross (†) for esophageal temperature. From Ref. 49; used with permission.

reduction in skin blood flow and sweating during inactive recovery, results in a time dependent transfer of heat from muscle to the core. Consequently, this contributes to a prolonged elevation in esophageal temperature (41). The redistribution of blood volume after exercise and its effect on esophageal temperature change has yet to be studied. However, convective heat transfer between muscle and blood is a major factor in the removal of residual heat content of muscle post-exercise. At this time, further studies are required to advance our understanding of the relationship between post-exercise tissue blood flow and changes in local tissue and whole-body heat content.

Altered skin blood flow and sweating responses: As previously noted, it has been shown that the heat loss responses of skin blood flow and sweating are altered during exercise recovery and are important factors contributing to the post-exercise sustained elevation of esophageal temperature (44). This is supported by studies showing that the magnitude of the increase in threshold has also been shown to vary as a function of exercise intensity (42,43) (Fig. 4 and 5). The threshold in this context is the esophageal temperature at the time when there is a sustained increase in cutaneous vascular conductance or sweating observed in three consecutive measurements. These studies suggested that dynamic exercise has a residual effect on the control of skin blood flow and sweating; however, it remains unclear whether these altered sudomotor and vascular responses are centrally mediated or whether these phenomena are peripheral in nature. For example, it is possible that dynamic exercise induces a local response at the level of the cutaneous vasculature or sweat gland. It is reasonable to infer that changes in these thermoregulatory effectors contribute to an attenuation of whole body heat loss and, therefore, to the prolonged elevation in esophageal temperature.

More recent studies suggest that the post-exercise sustained elevation in esophageal temperature is not due to a hypothalamic shift of set-point temperature, as

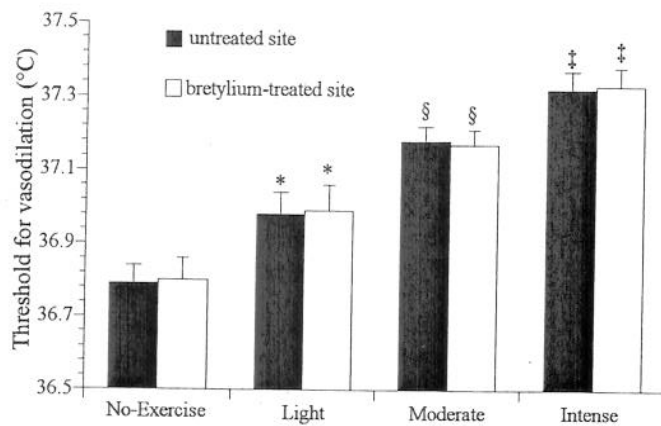


Fig. 4. Mean (\pm SE) onset esophageal temperature threshold values for forearm cutaneous vasodilation for no-exercise and post-exercise resting as measured following 15 min of exercise performed at 55 (light), 70 (moderate), and 85% (intense) of $\dot{V}O_{2peak}$ for the bretylium-treated and untreated forearm measurement sites. Exercise resulted in a significant increase in the threshold for cutaneous vasodilation above no-exercise resting (* $p < 0.05$). § indicates significant difference from the post-exercise - 55% $\dot{V}O_{2peak}$ ($p < 0.05$). ‡ indicates significant difference from post-exercise - 70% $\dot{V}O_{2peak}$ ($p < 0.05$). From Ref. 42; used with permission.

occurs during a febrile episode (67), but rather the result of nonthermal factors, such as those associated with blood pressure regulation (24,29,37,41). This hypothesis has evolved from the observation that an increase in the magnitude of post-exercise hypotension, induced by exercise of increasing intensity, was shown to: 1) result in a relative increase in the onset thresholds for sweating and cutaneous vasodilation; 2) cause an overall decrease in the rate of heat loss; and 3) cause a concomitant increase in the post-exercise esophageal temperature recovery time (41–43). Based on the observed relationship between the cardiovascular system and heat dissipation, it follows that factors determining post-exercise cardiovascular status may also attenuate post-exercise heat loss responses, and consequently affect temperature regulation. Before a discussion of possible nonthermal influences on post-exercise temperature regulation, a description of the typical hemodynamic responses post-exercise is warranted.

Hemodynamics Post-Exercise

There remains little information regarding the interaction of thermoregulatory and cardiovascular responses post-exercise. Given the pivotal role of the cardiovascular system in heat dissipation, it is essential to describe the changes that typically occur during exercise recovery. It is noteworthy that hemodynamic changes can be examined under early or late stage conditions. In the early stage, the greatest hemodynamic changes occur acutely after removal of the exercise stimulus and tend to be observed within the first 10 min post-exercise. The late stage, however, is sometimes typified by post-exercise hypotension (PEH), which is also of interest clinically in the management of hypertension. The occurrence of PEH is a prolonged response and can involve neurohumoral and vascular mechanisms between 10 min and 2 h post-exercise in healthy individuals. Studies conducted to examine the

early stage address different hypotheses than those which examine PEH. Early stage studies tend to look at factors affecting the acute hemodynamic changes after removal of the exercise stimulus such as baroreceptors, central command, and mechanoreceptors. Conversely, PEH studies tend to look at the cardiovascular mechanisms responsible for the prolonged decrement in mean arterial pressure (MAP). These studies tend to address autonomic and humoral changes over a longer time frame.

Early stage: During the initial phase of exercise recovery, there are dramatic changes in hemodynamic responses subsequent to a change in input from central command, mechanoreceptor, and metaboreceptor feedback. The hemodynamic changes observed are most pronounced in the first 5–10 min after exercise. At the cessation of dynamic exercise and during inactive upright recovery, MAP decreases rapidly during the first 1–2 min of recovery (6), as do stroke volume and cardiac output. In contrast, however, total peripheral resistance tends to increase to pre-exercise levels (6). Thoracic impedance values indicate a decrease in central blood volume during upright inactive recovery compared with that of active recovery modes (6,7). It is thought that decrements in central blood volume during upright inactive recovery are associated with the accumulation of blood in the venous system of the lower extremities in the absence of the muscle pump (6,19,46). Additionally, persistent muscle vasodilation (57) also contributes to lower central blood volume and thus reduced cardiac filling (46) in the upright posture. Despite a reduction in pre-load, it has been demonstrated that greater inotropic activity during the first 5–10 min of recovery can occur (47). The combined effects of warm blood stimulating the hypothalamus and sinoatrial node (18) and decreased atrial stretch result in an elevated heart rate. The outcome of the increased heart rate and contractility leads to a post-

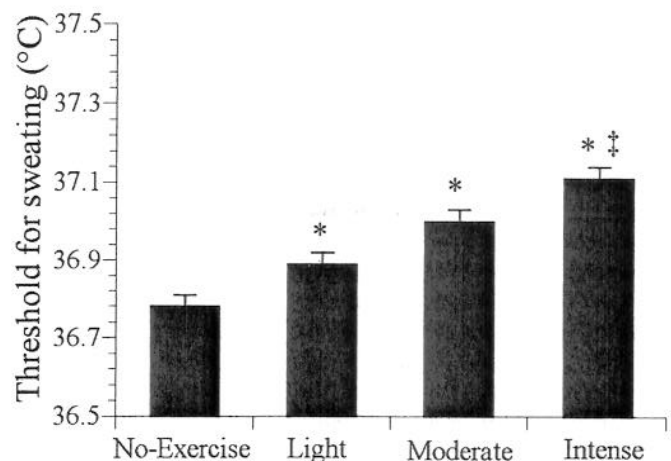


Fig. 5. Esophageal temperature at onset for sweating for no-exercise and post-exercise resting protocols as measured after 15 min of exercise performed at 55 (light), 70 (moderate), and 85% (intense) of $\dot{V}O_{2peak}$. Values are means \pm SE. Note: onset of sweating was measured a minimum of 30 min post-treatment (i.e., during the whole-body warming phase). Exercise resulted in a significant increase in the threshold for sweating above no-exercise resting (* $p < 0.05$). ‡ significant difference from light exercise, $p < 0.05$. From Ref. 43; used with permission.

exercise cardiac output that is actually equal to or exceeds pre-exercise values in the seated position (46).

Late stage: Although cardiac output can be equal to or greater than pre-exercise values, a deficit in peripheral resistance can lead to a decrease in MAP. A reduction in MAP after exercise was first described by Leonard Hill in 1898 (23), and subsequent studies have documented what is now considered the phenomenon of PEH (19,33). Post-exercise hypotension is thought to be caused by profound changes in the mechanisms that regulate and determine MAP (19–21). It should be noted that the magnitude of the reduction in MAP is greater with the orthostatic influence of upright or standing posture than when the subjects are supine (19,33). The level of MAP also appears to become significantly lower than pre-exercise values at approximately 20 min in both the supine (58,70) and upright seated postures (29).

Piepoli et al. (57) demonstrated there is persistent peripheral vasodilation post-exercise. This would tend to exacerbate pooling of blood in the previously active muscle when upright. Subsequently, work by Halliwill et al. (21) noted impaired sympathetic vascular regulation after exercise in the supine position and Pricher et al. (58) reported elevated leg vascular conductance up to 100 min post-exercise also in the supine posture. Thus, the combined effects of persistent neural and vascular adjustments as well as the upright seated posture with dependent lower extremities and no muscle pump results in significant pooling of blood (19,46). The decrease in cardiac filling during inactive recovery is sensed by cardiopulmonary baroreceptors. If there is significant post-exercise hypotension, then arterial baroreceptors may also be unloaded. It should be noted that in healthy normotensive individuals post-exercise hypotension is acute and temporary. Moderate intensity dynamic exercise for 30–60 min will produce reductions in MAP between 5–10 mmHg in the supine position that lasts several hours (19). Given the considerable cardiovascular adjustments described above, it is essential that the role of hemodynamic changes be assessed in the evaluation of post-exercise thermoregulation.

Non-Thermoregulatory Influences Post-Exercise

Changes in skin blood flow and sweating during exercise can be initiated centrally, but they are also subject to non-thermoregulatory controls such as central command, baroreflexes, muscle mechanoreceptors, and metaboreceptors (25,26,56,63). At the cessation of exercise, however, dramatic changes in MAP occur secondary to changes in input from central command, metaboreceptor, and mechanoreceptor feedback. Thus, skin blood flow and sweating may still be subject to non-thermoregulatory baroreceptor control during exercise recovery (7). This is plausible given that evidence exists for baroreceptor modulation of skin blood flow and sweating (11,27,32,48). Additionally, the post-exercise hemodynamic responses described above support the notion that during the post-exercise period, there is greater unloading of cardiopulmonary baroreceptors,

and possibly arterial baroreceptors depending on the degree of hypotension exhibited in the upright posture.

The interaction of baroreceptor reflexes and thermoregulatory effectors in the control of peripheral vasculature has been studied for many years (13) and continues to be well documented (9,25). Evidence also exists for non-thermoregulatory modulation of sweating (63); however, there is a relative lack of information regarding baroreceptor control of sweating and skin blood flow post-exercise. Jackson and Kenny (24) have observed a reduction in the post-exercise resting threshold for sweating and cutaneous vasodilation with the application of lower body positive pressure (LBPP). This shows that LBPP, which is known to reverse post-exercise hypotension and load baroreceptors, augments both vascular and sudomotor responses to subsequent heat stress. Kenny et al. (42,43) have also demonstrated that the post-exercise onset thresholds for sweating and cutaneous vasodilation are increased in parallel with the magnitude of post-exercise hypotension observed (Fig. 4 & 5). That is, the greater the post-exercise hypotensive response, the more the thresholds are delayed. Furthermore, by employing the iontophoretic application of bretylium to one skin site, they showed that the increase in threshold for cutaneous vasodilation was not due to enhanced adrenergic vasoconstrictor activity (Fig. 4), but rather due to an alteration in active vasodilation. This research implies a possible graded relationship between the magnitude of hypotension measured and the magnitude of change in sudomotor and vascular control as indicated by a change in onset threshold.

While the data regarding baroreceptor influence on the cutaneous circulation is consistent, sweating data is less accordant. Under resting conditions, some researchers did not observe decreases in sweating with baroreceptor unloading (69,74) while others have observed a reduction in the thermosensitivity with baroreceptor unloading at rest (66) and during exercise (28,48). There remains conflicting information regarding the possible baroreceptor influence on sweating during the post-exercise period. More recently, studies support the concept of a baroreceptor-mediated influence on sweating (24,29,30,43); however, this controversy has yet to be resolved.

In order to determine the relative nonthermal contributions to skin blood flow and sweating during exercise recovery, a series of studies were proposed using recovery mode as a variable to control for nonthermal inputs (7,30,64,73). The principle of using the modes of active, inactive, and passive recovery in the study design was to delineate the relative roles of central command, mechanoreceptors/skeletal muscle pump, and baroreceptors in the modulation of sweating and skin blood flow. Subsequently they have been used to study the nonthermal influences on heat loss responses. The mechanisms of the different recovery modalities are: 1) during active recovery (loadless pedaling), skeletal muscle pump/mechanoreceptors and central command are activated; 2) during passive cycling, mechanoreceptors are stimulated without the involvement of central command (6,54,55,72); and 3) during inactive recovery,

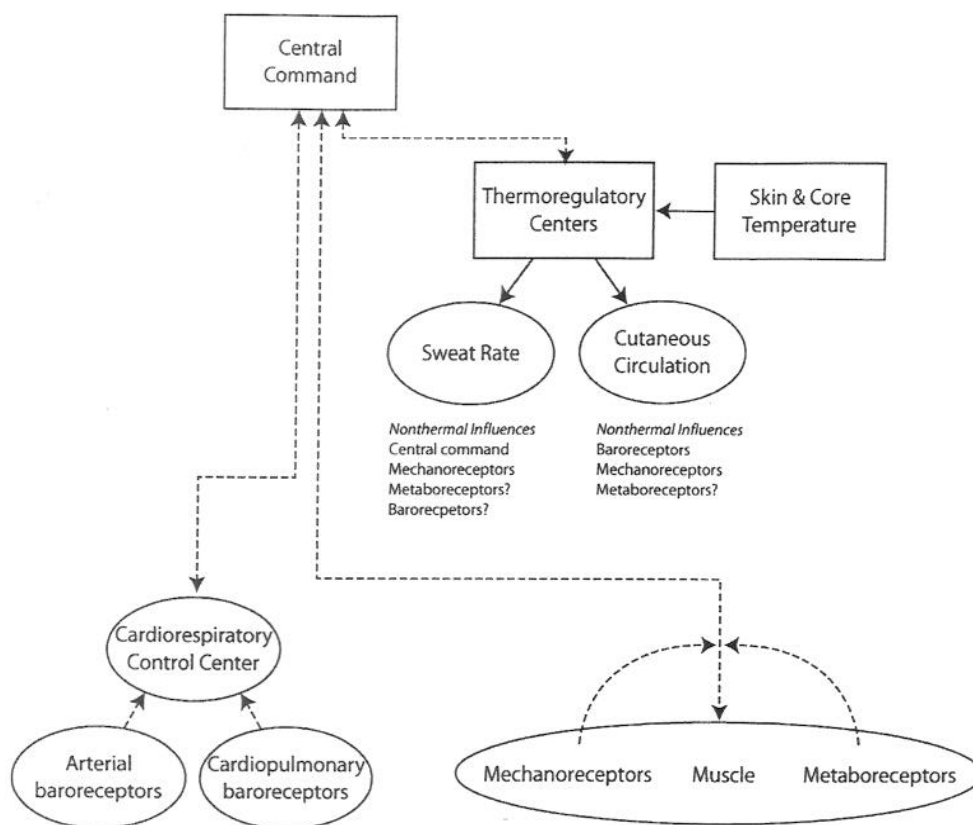


Fig. 6. Schematic summary of non-thermal influences on post-exercise heat loss responses. The figure presents our current understanding of these influences as concluded by the recovery mode studies discussed in the text. These studies were conducted in thermoneutral conditions using short-duration moderate-intensity exercise. The '?' denoted means that the influence listed remains to be studied or is still unresolved based on the research presented in this review. Solid lines indicate known thermal influences on sweating and the cutaneous circulation. Dashed lines indicate nonthermal inputs.

baroreceptors are primarily implicated. While baroreceptors are implicated in each recovery mode, it is believed they are the primary nonthermal influence on skin blood flow and sweating in the inactive mode (7,29,39). Thus, the relative effects of active, passive, and inactive modes, and by extension the roles of central command, skeletal muscle pump/mechanoreceptors, and baroreceptors, can be evaluated. Taken together, the studies suggest that attenuating the baroreceptor unloading effect associated with upright inactive recovery through an active or passive recovery mode preserves skin blood flow. Post-exercise measurement of sweat rate responses indicate that sweating can be modulated by central command and mechanoreceptors. One of the studies (30) showed a separation in the MAP responses between recovery modes when in the upright position, which, therefore, cannot exclude the role of baroreceptors in the modulation of sweat rate. It is unclear why the separation in MAP was observed. Aside from employing greater exercise intensity than other investigations, the study used a similar protocol and posture to other studies. A summary of the non-thermal influences on heat loss responses as concluded by recovery mode studies is displayed in the schematic in Fig. 6.

While these studies have demonstrated that both active and passive recovery modes can preserve skin blood flow and sweating responses, it remains to be determined whether this response can significantly affect esophageal temperature reduction in a prolonged recovery. Journey et al. (30) and Shibasaki et al. (64) employed recovery modes of 15 min and 20 min, respectively, and did not report significant dif-

ferences in esophageal temperature between modes. In keeping with the concept that a relationship exists between heat loss responses and hemodynamic status post-exercise, a recent study by Journey et al. (29) showed that the application of LBPP post-exercise resulted in a reflex increase in skin blood flow, sweating and heat flux, and resulted in a more rapid decay in esophageal temperature relative to the same values measured under lower body negative pressure and no pressure conditions. They postulated that the LBPP attenuated the baroreceptor unloading effect of seated recovery through a shifting of blood to the central circulation from the lower extremities. The increase in central blood volume secondary to LBPP is thought to have triggered a baroreceptor-mediated increase in skin blood flow and sweating and thus heat flux, leading to the observed accelerated decline in esophageal temperature. This is further supported by earlier observations by Jackson and Kenny (24) that application of LBPP increased the rate of esophageal temperature decay. Recent work by McInnis et al. (49) further supports a baroreceptor-mediated mechanism in the post-exercise attenuation in heat loss. In their study they applied 15° head-down tilt to subjects immediately post-exercise, which attenuated the fall in MAP, cutaneous vascular conductance, and sweat rate, and resulted in an increased rate of esophageal temperature decay throughout recovery. The head-down tilt intervention also shifted the esophageal temperature relationship with cutaneous vascular conductance and sweat rate as compared with the upright, seated recovery posture (Fig. 2 & 3).

Gender and Post-Exercise

The current understanding of post-exercise temperature regulation contains some inherent bias such that only male subjects have been studied or not enough female subjects were included to determine possible gender differences in post-exercise thermoregulation. Given the apparent correlation between post-exercise cardiovascular regulation and thermal responses, it is important to examine the effect of gender in light of different recovery responses. Carter et al. (5) suggested that women may be more susceptible to post-exercise orthostatic hypotension and that active recovery should reduce that risk. They observed a greater reduction in MAP and less compensatory vasoconstriction in women than in men. In their study, however, the participants performed only 3 min of low-intensity exercise. Other studies have also shown a greater magnitude of post-exercise hypotension in women compared with men in the upright posture (15,40,62). Fisher et al. (15) demonstrated a gender difference in post-exercise MAP measured over the course of a 90-min recovery following 35 min of cycling at 60% $\dot{V}O_{2peak}$. In addition, Senitko et al. (62) showed a gender difference in post-exercise MAP in the upright position in sedentary individuals following cycling at 60% $\dot{V}O_{2peak}$ for 60 min. In another study, it was shown that compared with men, women demonstrated a greater magnitude of post-exercise hypotension and this was accompanied by a greater elevation in the post-exercise threshold for cutaneous active vasodilation (40). The subjects in these studies were tested in the follicular phase of the menstrual cycle to control for possible hormonal influences.

While the effect of recovery mode, and by extension the relative nonthermal contributions to post-exercise cutaneous vascular conductance and sweating, have been described in men earlier in this review, a study has shown that women demonstrate a difference in the control of cutaneous vascular conductance during exercise recovery (31). Therefore, while men and women share the nonthermal mechanoreceptor and baroreceptor influences on cutaneous vascular conductance, they differ in the nonthermal contribution of central command in women. Nonthermal control of sweat rate appears similar between genders. Thus, the effect of gender differences in cardiovascular regulation should be examined in the context of the possible consequences to post-exercise heat loss.

Hypohydration Post-Exercise

Body water deficits (hypohydration) are associated with increased cardiovascular strain (52,53,60) and under very severe conditions possibly cardiac dysrhythmia (61) and perhaps cardiac arrest (8). Hypohydration mediated cardiovascular strain results primarily from reduced plasma volume. Under hyperthermic conditions, this hypohydration mediated cardiovascular strain can be exacerbated due to redistribution of blood to warm skin (61). Also, passive hyperthermia alone (which is augmented by exercise and hypohydration) is reported to decrease cardiac vagal modulation of heart rate (12). Despite the well-characterized effects of hy-

pohydration on cardiovascular and heat strain, its influence on post-exercise thermoregulation is generally unknown.

Both during and after exercise heat stress, hypohydrated subjects can have altered muscle metabolism (14), reduced baroreceptor responsiveness (10), difficulty sustaining BP (17), elevated circulating catecholamines (51), greater hyperthermia (61), and impeded physiological adaptations to heat acclimation (3). Each of these factors may influence post-exercise thermoregulation and subsequent exercise bouts. Repeated bouts or days of prolonged low to moderate intensity exercise in a hot, humid environment represent a practical scenario for many occupations. Although intensive research has examined the relationship of hydration status and thermoregulatory and cardiovascular responses during exercise, data are very limited with regards to the post-exercise period.

Recently, the effects of hypohydration (4% body-weight loss) on post-exercise seated recovery in the heat (40°C, 20% relative humidity, 1 m · s⁻¹ wind speed) was examined (4). When the subjects initiated the hypohydration exercise-heat trial, they had been dehydrated ~15 h before and had rested quietly in a temperate climate (~24°C, ~45% relative humidity). It was shown during 45 min of exercise recovery while hypohydrated in the heat (40°C), seated heart rate and esophageal temperature remained elevated relative to the euhydration condition (normal body water balance).

Charkoudian and colleagues recently examined the influence of hypohydration (1.6% body mass loss) on cardiovascular control after exercise-heat stress (10). A nitroprusside and phenylephrine challenge was used to examine BP responses and baroreceptor sensitivity 90 min after completing exercise. The nitroprusside and phenylephrine challenge is a commonly used pharmacological approach to induce acute, rapid changes in BP to assess baroreceptor sensitivity. When subjects were hypohydrated by 1.6% bodyweight loss, cardiac baroreceptor sensitivity was reduced compared with when euhydrated. Charkoudian and colleagues showed that restoration of plasma volume by saline infusion (rehydration) does not immediately restore these physiological responses after exercise-heat stress (10). Data from Charkoudian et al. (10) demonstrate that BP regulation may be profoundly influenced by thermal stress via altered baroreceptor mechanisms after exercise. However, more data are needed to clarify mechanisms related to altered thermoregulatory and cardiovascular responses during the post-exercise period when hypohydrated. In addition, studies should address these physiological responses after exercise across temperate and hot environments.

Summary

Thermoregulation during the recovery period from exercise is affected by many factors. Current data suggest that non-thermoregulatory influences associated with hemodynamic regulation and hydration may have a prominent role. This is supported particularly by the apparent link between the perturbed thermal responses and the concurrent cardiovascular changes incurred af-

ter a bout of dynamic exercise. Further research is needed to address how these thermoregulatory and cardiovascular responses interact and ultimately regulate body temperature after exercise.

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